# Original Articles.

## CASES OF MULTIPLE NEURITIS.[1]

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ALTHOUGH primary, multiple, degenerative neuritis has been recognized and described for twenty years or more, very little attention has been given to the disease until within five years. The first cases fully recognized in Boston, or, at least, reported, so far as I know, were four years ago, when there was quite a number of idiopathic cases in this city, and the disease prevailed also in several stables, among horses.

Among the cases which I have seen there have been three types: (1) Idiopathic, in regard to which the opinion now prevailing is that the disease is of infective origin, analogous to beri-beri. (2) Toxic. (3) Purely rheumatic, like Bell's palsy. I will describe only a few typical cases, alluding briefly to others.

C. R., a Swede, thirty-eight years old, married, a rigger and sailor, entered the Boston City Hospital, in my service, July 6, 1886. A week previous he was on deck, in an exciting yacht race, in his stocking-feet, and got his feet and legs thoroughly wet. In the evening he thought that he had taken cold. The next day his legs felt heavy, and he could not go aloft, but worked on deck. The following day he went home and took to his bed, complaining of severe pain in the small of the back, knees, ankles, and feet, moderate headache, loss of appetite, nausea, and excessive thirst. He had, also, a slight chill. He complained of a dead feeling in his legs, and said that he could not walk. He could not sleep from pain in his limbs and joints, and restlessness. Two days after the trouble in his legs his hands felt numb, and his arms were observed to be weak; and still two days later, he began to have difficulty in drinking, on account of inability to tightly close his mouth. He thought that his speech was altered, being somewhat thick. He had perspired excessively.

The patient was a strong, well-developed, and well-nourished man, without ascertainable hereditary predisposition to disease, a moderate drinker, and, according to his statement, without a previous history of syphilis.

On examination, there was no evidence of disease of the thoracic, abdominal, or pelvic organs, nor of the brain and spinal cord. Temperature was 99.4°, pulse 96, respiration 32. Tongue slightly coated, moist; protruded straight, but only as far as about one-quarter of an inch beyond the line of the teeth. The eyelids failed to close by about the space of one-half an inch, and the lips by one-quarter of an inch. No affection of the motor muscles of the eyeballs; pupils not abnormal. Speech decidedly thick, cheeks puffed out a little, eyebrows cannot be raised. The grasps of both hands were weakened and nearly equal. The arms could be moved freely, but not used. The legs could be moved only very slightly.

There was some sensation of prickling, very slight, in the feet, and a numb or "dead" feeling in hands and legs. Superficial sensation was somewhat impaired in the feet, and very slightly in the legs; elsewhere unimpaired, except for some cutaneous hyperæsthesia in thighs, arms, trunk, and face. No difficulty in respiration. The disease was very nearly symmetrical; if any difference, the left side was slightly more affected than the right.

The urine was normal, of specific gravity 1026. The bowels were constipated, micturition normal, spleen not manifestly enlarged; no pain or swelling in any joints. On lying still in bed, there was no pain anywhere, but motion of the legs was painful, and pressure, even moderate, over the nerve-trunks, and in the course of the nerves in the legs and thighs, gave rise to exquisite pain. There was no evident muscular atrophy. The chin- arm- and knee-jerks were absent, and there was no response from the plantar, epigastric, and abdominal muscles; very slight from the cremasteric.

In the course of the disease, the pain became so extreme that subcutaneous injections of morphia and laudanum externally were used freely for three months. The tenderness on pressure over the course of the nerves, from the shoulders down, became everywhere excessive. There was also great pain in opening and closing the jaws, and tenderness on pressure over the motor branch of the fifth pair. The paralysis gradually reached a point where the patient could not turn in bed from his back to his side, or move his legs. The fingers closed only faintly in the motion of grasping. The wrist-drop was not very marked. The paralysis of the extensors of the feet was absolute, so as to require the use of foot-splints. There was extreme loss of flesh in the legs, and generally considerable quantitative lowering of electro-muscular contractility, and, finally, lack of response in extensor muscles and some others to either current, faradic or galvanic. The bowels became most obstinately constipated. There was troublesome retention of urine, but not sufficient to require the catheter. There was also a small bed-sore.

The mental condition was of a mild stupor, approaching delirium, to the extent that, for a few weeks, the patient's statements with regard to anything which had happened several hours previously could not be depended upon. The aspect was distinctly typhoidal. The spleen was not found to be enlarged at any time. A very mild degree of anæsthesia, just noticeable, extended, in time, over the thighs, trunk, and arms, and it increased in the legs and feet.

At the end of the first month the tongue was nearly clean, and slight improvement was observed in the hands and face.

At the end of the second month, the patient could move his arms pretty well and close his fingers in a grasp, but not exert force. He had very little trouble in eating, as he could close his lips, and there was no pain in chewing. There was some tenderness on pressure over the motor branch of the fifth pair, but scarcely any over any of the nerves of the arms, chest, trunk, abdomen, or thighs, but excessive over those of the legs. He could almost close his eyes, and could move from side to side, but not sit up. There was no soreness on motion, but the pain in the legs still required opiates, especially at night. The flexor muscles of the forearm reacted to the faradic current; the extensors, the quadriceps femoris, and all of the leg to neither current. Massage was begun, and, three weeks later, galvanism, beginning where extreme tenderness on pressure had disappeared, galvanism following massage, until both were gradually extended over the whole body.

[1] Read before the Section for Clinical Medicine, Pathology and Hygiene, of the Suffolk District Medical Society, April 13, 1887.

At the end of the third month, marked and steady improvement had been observed everywhere, except in the legs. There was complete ankle-drop; and the muscles of the thighs and arms, but especially of the legs, were very much wasted, soft, and flabby. There was still great tenderness on pressure over the calves, but there seemed to be no well-marked diminution of sensation, except in the feet, and that not very great. Two weeks later he was sitting up daily, and in another week there was scarcely any tenderness anywhere on pressure in feet or legs.

A little after the close of the fifth month, the foot and toes were seen to move, for the first time, on the right side, and the tibialis anticus and gastrocnemius muscles contracted. In a few days, similar observations were made of the left foot and toes; but, at the end of the sixth month, he was still unable to stand. A week later he began to walk, with support, and two weeks after that he could walk alone. Two weeks later still, he was discharged at his own request, that is, about a week after the seventh month of treatment. All the muscles, at that time, reacted to galvanism, although in degree considerably below the normal in the legs and feet, less so in the arms. He could raise his feet and toes, although they dropped somewhat in walking.

At the end of the ninth month he had regained his usual flesh, and, except in his legs, he said, his usual strength. He could walk perfectly well on a level, but, going up or down stairs, or over curbstones, needed a cane or some similar support. I found him, after some difficulty, on the street, and could not make a full examination. The knee-jerks were still absent, but the patient expects to go to work soon.

In accordance with my previous experience in such cases, I found very little benefit from salicylic acid or the salicylates in treatment, but the pain always diminished after using quinine in full doses.

As regards diagnosis, the muscular wasting and electrical reactions exclude Landry's paralysis, which is, I believe, now accepted to be a rapidly fatal form of myelitis; the gradual invasion of one group of muscles after another, with sensory disturbances, and the pain and tenderness on pressure, exclude anterior poliomyelitis, and no other diseases would suggest themselves. After admission to the hospital, the temperature did not exceed 100° F., nor the pulse 102. But the febrile symptoms, and typhoidal appearance and mental condition, place the case among the infective diseases, and, therefore, in the first group of idiopathic, primary, multiple, degenerative neuritis.

Although there was a previous history of exposure to wet and cold in this case, there has not been such in my other cases. Judging from previous experience, the prognosis in this case seems to me favorable for the reappearance of the knee-jerk and ultimate greater improvement, if not entire restoration of function in the legs, but probably not for some months, and possibly, not within a year or two.

I have not been able to make out any distinct enlargement or hardness of any of the nerves in these cases. Indeed, the exquisite pain prevented an examination careful enough to settle these points.

Of the toxic cases, I am not sure that I have ever seen arsenical multiple neuritis. At least, the diagnosis was not confirmed, or otherwise, by chemical examinations of the wall-paper or urine. Those due to lead are readily diagnosticated by the usual methods of testing for its presence in the urine. A previous history of syphilis, especially after a rapid relief by mercury or iodide of potassium, of course, justifies the assumption that the case is syphilitic. Cases after the infective-diseases, especially typhoid fever and diphtheria, are reported, and have been probably seen by most of us. In the course of chronic disease of the lungs and kidneys, especially in the late stages, they are not very uncommon. One of the most distressing forms of multiple or general neuritis, and perhaps the most hopeless, is due to the long-continued abuse of the preparations of opium. Where it exists the abandonment of the opium habit and its continuance are so nearly equally painful and fatal that there is little choice between them. By far the most frequent source of toxic general neuritis, however, is alcohol; and cases occur in all degrees of severity, of which I shall report two, one at each extreme.

Mr. ——, a literary gentleman, just beyond middle age, born in Europe, came to this country with the habit established of drinking a pint of claret with his dinner. Under the press of heavy work, he increased his pint of claret to a quart, and drank a half-tumbler of whiskey at bed-time. He also used tobacco freely.

On account of some failure in eyesight, not excessive, impaired facility of using the legs, which was troublesome in going up and down stairs, etc., but otherwise not considerable, a dull pain in the thighs and legs, disappearance of the knee-jerks, and marked swaying of the body with the eyes closed, the confident diagnosis of locomotor ataxia had been made. There were some slight anæsthesia and paræsthesia. I could not find any symptoms that seemed incapable of explanation from the alcoholic history, although the patient had never been intoxicated in his life and had never been conscious of taking alcohol to excess. There was some, though slight, tenderness on pressure over the course of the nerves in the legs. The alcoholic habit was given up; in a year the knee-jerk had re-appeared and the symptoms suggesting the diagnosis of posterior spinal sclerosis were gone. This mistake is, I think, a not very infrequent one, and occurs, too, in multiple neuritis due to syphilis or lead. I have also seen cases of locomotor ataxia where there seemed to me to be symmetrical peripheral neuritis, to the relief of which I attributed great temporary improvement in the symptoms.

M. H., single, twenty-six years old, employed in a bar-room, entered the Boston City Hospital, August 12, 1886. His father died of rheumatic fever. Eight years previous to admission patient had typhoid fever and once fell unconscious in a fit after drinking heavily. He had been a hard drinker for many years, and for a week past had been sleepless and without appetite, drinking steadily. After sleeping in a cellar, drunk, he awoke with severe pains in his head, back, and entire left side.

On entrance he appeared well developed, and fairly nourished. Temperature 100.4°, pulse 104, respiration 28. Tongue rather dry, slightly coated. Bowels constipated. Urine, a trace of albumen. The examination of thoracic, abdominal and pelvic organs was negative.

Pupils of moderate size, right somewhat larger, both react fairly well; marked nystagmus on looking to either side; motion of left eye outward somewhat limited. Some tremor of fingers, left grasp weaker than right. Coördination good. Sensation unim-

paired in head, arms and trunk. In both legs below knee are one or two areas of diminished sensation. Left knee-jerk nearly, if not quite absent; right very weak. Superficial reflexes well marked. Slight tenderness along nerve-trunks in both legs and right thigh, possibly somewhat in forearms. Mental condition not remarkable.

13th. Increase of pain in arms, legs, chest and abdomen. Marked tenderness over course of nerves in legs, arms and intercostal spaces. Complains of numbness in hands, but there is no diminution of sensation. Both grasps distinctly weak. Considerable loss of power in extensors of feet and toes. Knee-jerks absent.

14th. Last night became very delirious and actively violent, requiring restraint, and during which he became unconscious of pain or tenderness. Somewhat quieter this morning, but still delirious and constantly talking. Excessive muscular tremor. Takes nourishment fairly well.

15th. In a mild semi-delirious state, picking clothes, etc.; very weak, with feeble pulse. Extreme sensitiveness to pressure over course of nerves everywhere but in the head and neck; screams with pain with the least touch or movement of the body and limbs.

18th. Is still quite delirious, complains of soreness and pain all over; marked anæsthesia especially in legs.

21st. Marked loss of power in legs and some in arms. Toes drop. Over both buttocks are abrasions of skin, superficial, size of dollar. Urine and fæces passed in bed. Mental condition quite suggestive of the final stage of general paralysis.

25th. Loss of power in arms and legs seems greater.

28th. Complete wrist- and toe-drop. Is more delirious, the delirium frequently being of a tearful and painful character; often shouts in imaginary conversation. Eats well. Extreme sensitiveness on pressure over the nerves; muscles very flabby, with very little power of movement in muscles of arms, legs and trunks.

September 2d. Constant hallucinations of sight and hearing. Has no idea where he is, and most of the time recognizes no one about him; has been seen to put fæces in his mouth. Cannot turn over in bed without help.

5th. Bed-sore over right buttock, irregular in outline, superficial, about two inches in diameter. Small abrasion over left buttock. Habits very filthy. Distinct nystagmus noted at times. Sleeps but little.

8th. Can now turn himself in bed and feebly extend right wrist. Mental condition no better. Sleeps poorly. Takes any food given him.

18th. Is delirious all the time when awake, and thinks he spends his time in the woods, down by the wharves, and on long walks in the country. Last night restless and noisy, and rolled out of bed. Cannot walk on hands and knees.

23d. Less noisy. Bed-sores improving.

29th. In general, seems to be improving somewhat.

October 6th. Is able to extend right wrist slightly. Grasps very weak, but growing stronger. Still marked sensitiveness over nerve-trunks, especially in legs. Feet completely dropped, but there is some power of voluntary motion in toes. Very marked general wasting; flabbiness of muscles. Mental condition decidedly better; eats and sleeps well without opiates. Can turn in bed. Anæsthesia very moderate and chiefly in the feet and lower part of legs.

13th. Quieter and more rational; eats and sleeps well.

17th. Memory extremely poor, but shows no delusions.

22d. In general, is improving physically and mentally, and in good spirits.

27th. Gaining both in motion and sensation of extremities. Tenderness over nerve-trunks much less.

31st. Still some tenderness over nerve-trunks. No evident impaired sensation. Knee-jerks absent. Feet and toes dropped, but both can be moved and raised a little. Grasps weak but about equal. Elbow-jerks present. Muscles of limbs thin and very flabby.

November 24th. Gradually gaining; still considerable tenderness in legs and pain on motion. Some motion in both feet and toes.

December 21th. Improvement continues; can walk a few steps without assistance.

January 10th. Quite comfortable; complaining of some pain in lumbar region and left hip. Otherwise pain and tenderness on pressure gone.

16th. Improving rapidly, is up and dressed daily, and can walk quite well. Muscles react fairly well to galvanism.

18th. Discharged at his own request, and I have not been able to learn anything of him since that time.

The treatment in this case consisted of rest in bed, abundant food, opiates very freely, the actual cautery and small blisters over the course of the most painful nerves, and, later, after the soreness on pressure had nearly disappeared, massage and galvanism. The reaction to both currents in this case was decidedly diminished, but the reaction of degeneration was not found.

As illustrations of what I supposed to be purely rheumatic cases, and, perhaps, throwing some light on the question of pathology, I will very briefly report two cases:

The first was of a young man, who got drunk, lost his way, and slept in the woods in the snow, his shoulders having been particularly exposed. On awaking, he had severe pain in his shoulders and arms, which gradually increased, so as to become most intense, and increased by motion of the arms, with tingling and numbness in the hand and fingers. There was almost entire paralysis when I saw him, a few days later, diminished reaction to the faradic current, and extreme tenderness on pressure over the nerve-trunks. With rest, warm anodyne fomentations, quinine in large doses, and later, rubbing and electricity, there was complete recovery in six weeks.

The second was a young carpenter in Newport, kindly sent to me by Dr. Engs, of that city. After working all the afternoon, wet through in a cold rain, he spent the evening with his wet shirt on, after having changed his other clothing. The next morning he had severe pain in his arms, which soon increased to a degree which was quite excruciating, and lasted for several weeks. Whether there were other sensory symptoms or not then, I could not ascertain: I saw him three months later, and found marked atrophy and extremely limited power of motion in both shoulders and arms. The pain had subsided so as not to be especially troublesome, and there was not marked tenderness on pressure over the nerve-trunks. There was some reaction to the faradic current, and no marked sensory disturbance, that is to say, inflammation had subsided, and processes of recovery or of degeneration were to begin.

At the time of the examination, the patient's condition might have been explained by a diagnosis of an-

terior poliomyelitis; but, with the history, considering it a case of neuritis, I gave a favorable prognosis. He was sent to the Massachusetts General Hospital for convenience of treatment, and in three months returned home somewhat improved. He gained steadily at home, and when I saw him, a year later, there was scarcely any atrophy of the arms, and he had very good use of them. His hands were still not sufficiently recovered to resume his trade, although he could use them freely in eating, dressing, and plain work. The extensors of the hand were mostly at fault, but as they were capable of contraction and some usefulness, I gave as a prognosis that, in a couple of years more, the patient would be likely to have quite useful hands, so as to be able probably to resume his trade.

The pathology of these various cases has not yet been so far investigated as to place it on an entirely substantial basis. That they have many features in common is evident.

So far as I am able to learn, post-mortem examinations in cases of primary multiple neuritis have not thus far shown disease of the brain or spinal cord, which has not been acknowledged to be insufficient to produce the symptoms, and in most cases there has been no central lesion found; the evidence being that the disease is primarily an interstitial peripheral neuritis, that in mild cases the disease goes no farther, and that in severe cases there is also parenchymatous inflammation, and more or less degeneration of nerve-fibres. Both processes are capable, in time, of a great degree of regeneration, or, at least, of restoration of function. The distinct sensory symptoms with which the disease is commonly ushered in usually soon nearly disappear, except pain and marked anæsthesia is a rare exception in the disease. We are driven, therefore, to suppose (1) that there is an undiscovered central lesion; (2) that there is a functional central disease giving rise to the neuritis, as Erb holds; (3) that Leyden and Oppenheim are right that the central lesions thus far observed result from ascending neuritis, or form a part of the morbid process in the nerves, without giving rise in themselves to any special symptoms; or finally (4), with Strümpell, that primary multiple neuritis affects chiefly the motor fibres — a supposition analogous to Westphal's theory that the motor fibres or cells in the cord may be sensibly diseased without affecting those governing nutrition of the muscles.

Alcohol has an affinity, so to speak, whether from a common microörganism or not, for the brain and spinal cord, as well as for the nerves, and clinical evidence, supported by a certain number of autopsies, supports the theory that many, at least, of the cases of alcoholic and lead neuritis are complicated with more or less cerebral or spinal disorder, or both. That a central lesion, cerebral or spinal, may not ultimately be found to be a necessary factor in the idiopathic and other forms of the disease, of course, I am not prepared to say.

There are cases, also, doubtless, which are primarily subacute or chronic parenchymatous degeneration of the nerve-fibres, without marked symptoms, except loss of power, or impaired sensation. Perineuritis also occurs, and there are mixed cases.

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There are now four cases of leprosy in Minnesota as against six in 1884.

## FOUR HOSPITAL CASES.[1]

I. TUBERCULAR PERITONITIS, WITH PERFORATION OF THE ABDOMINAL WALL. II. CIRRHOSIS OF THE LIVER. III. HÆMOPHILIA. IV. TETANY.

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### I. TUBERCULAR PERITONITIS; SLOUGHING OF THE UMBILICUS AND FISTULA.

P., TWENTY-FOUR years of age, entered the hospital, June 21st. A sister died of phthisis. About a year before entrance the patient began to suffer from attacks of abdominal pain, distension and tenderness, lasting five to ten hours, and relieved by bilious vomiting. These attacks recurred at intervals of about a month. The last was four weeks before entrance; he did not rally from it but felt poorly, lost flesh and strength, and had moderate diarrhœa much of the time. He had no cough or other pulmonary symptoms. Pulse 100.

Physical examination showed slight consolidation at the right apex without softening. In the umbilical region a rounded cake-like tumor with irregular surface, four inches in diameter, and apparently covered over by intestine, was felt. On the right side of the tumor and in the right iliac fossa, tenderness was marked. Diagnosis: tubercular peritonitis.

July 3d. The patient was evidently weaker. Moderate fever was constant with evening exacerbations. The abdomen was more distended, peritoneal crepitus could be felt; night sweats, occasional vomiting, and abdominal pain were noted. The scanty sputum was examined for bacilli with a negative result.

July 14th. Several days before this date it was noticed that the skin about the navel was getting red and œdematous. On this date a small perforation took place through which with each inspiration was emitted offensive gas, on deep inspiration offensive greenish fluid; during the act of vomiting this fluid was ejected with force.

July 18th. The fistula gradually enlarged, and the patient was transferred to the surgical side.

July 19th and again on the 22d, dejections of normal consistency passed the rectum, the only discharges of any kind through that outlet between the appearance of the perforation and death, which took place July 28th, from exhaustion.

*Autopsy.* The right pleural cavity was obliterated by old adhesions, a few of which were also found on the left side. The apex of the right lung was thickened, shriveled and dense; on section, numerous small gray tubercles, and some pigmented fibrous tissue were seen. Throughout both lungs there were occasional cheesy patches, a half-inch or more in diameter, some of them partially softened, surrounded by deeply injected borders containing miliary tubercles.

A fistulous opening through the umbilicus contained a drainage-tube entering the peritoneal cavity partially obliterated by old adhesions. This encysted cavity extended upwards beneath the right lobe of the liver, and downward on the right to the pelvis, containing masses of necrotic fat tissue (omentum), and several ounces of offensive fluid; it communicated with the rectum above the internal sphincter by an opening in the anterior wall half-an-inch in diameter. The intestines contained tubercular nodules and ulcerations, chiefly in Peyer's patches.

[1] Read before the Section for Clinical Medicine, Pathology and Hygiene, of the Suffolk District Medical Society, April 10, 1887.